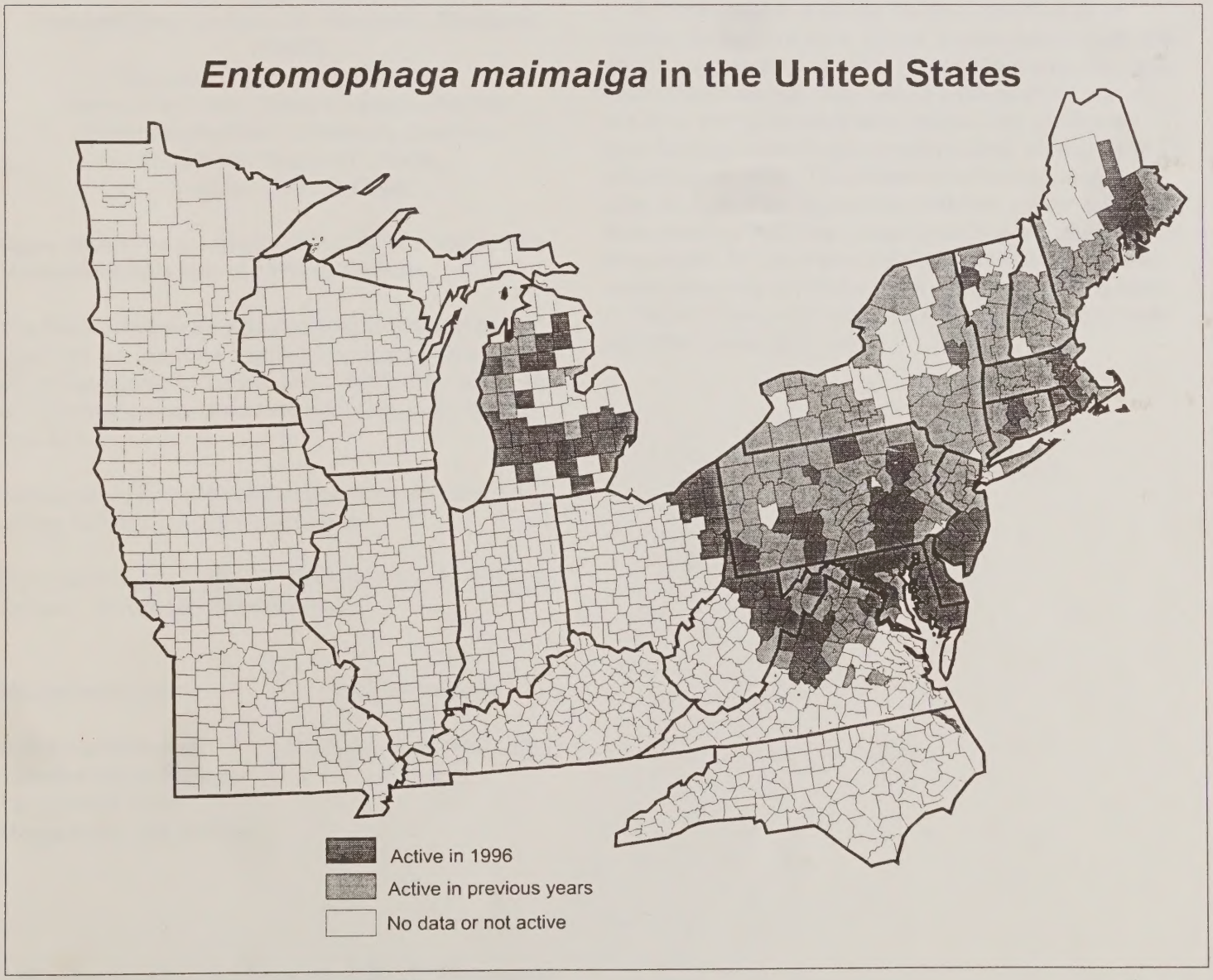
Gypsy Moth News

December 1996 Issue Number 42

Outbreak of 1996--
But it's not gypsy moth!



FOREST SERVICE
U.S. DEPARTMENT OF AGRICULTURE

NORTHEASTERN AREA
State and Private Forestry

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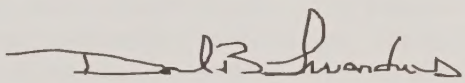
Editor's View

The map on the front cover, which in past years would have shown a county by county depiction of gypsy moth outbreak, now shows where gypsy moth isn't. *E. maimaiga*, the fungus that ate the gypsy moth, is in outbreak status throughout much of the Northeast.

The unanswered question is, what long-term effect will the fungus, *E. maimaiga,* have upon gypsy moth populations? We know that in most of the northeastern United States, the fungus has dramatically reduced gypsy moth numbers. Experts differ in their views. Some have said the gypsy moth collapse is merely the lucky combination of weather, fungus, and virus. Others propose that because of the fungus, we have seen an end to the periodic buildups in gypsy moth populations that once claimed millions of forest acres. While we wait to see what does happen, State and Federal agencies responsible for gypsy moth control, must adapt to life without gypsy moth. The dilemma for State agencies, which carry the bulk of the suppression workload, is how to maintain an experienced staff long enough to see if gypsy moth populations return. No one wants to reduce staff or disassemble an organization only to find the gypsy moth come roaring back. At the same time, what to do with a room full of gypsy moth expertise, and no gypsy moth?



Gypsy Moth Populations Plummet in 1996 While "The Fungus" Skyrockets

Noel F. Schneeberger

Time will tell if 1996 becomes a watershed year for gypsy moth in the United States. In total, gypsy moth defoliation was the lowest on record since 1968. Defoliation declined more than 85 percent from 1995 levels, to a low of 202,472 acres scattered across 11 States. As shown in the table below, States in which defoliation declined far exceeded those with increases. One of the most dramatic declines occurred in Virginia. Other States experienced similar declines albeit, not of the magnitude observed in Virginia.

Gypsy moth defoliated acres, 1995-1996

State	1995	1996
CT	2,704	1,404
DE	65,462	534
MA	8,707	9,274
MD	93,864	11,148
ME	500	100
MI	85,907	3,231
NH	1,700	210
NJ	39,580	27,990
NY	200	16,285
OH	34,405	49,448
PA	132,487	9,027
RI	50	0
VA	850,000	0
WV	102,971	70,726
Total	1,418,537	199,377

It appears that *Entomophaga maimaiga*, a fungal pathogen of the gypsy moth first observed in New England in 1989, has spread throughout much of the area considered to be generally infested by gypsy moth (see map on front cover). The only exception may be the transition area into which the insect is spreading for the first time. Increased defoliation in States, such as Ohio, suggest that the fungus may not have caught up to the advancing front of gypsy moth populations in newly infested areas.

Gypsy moth suppression and eradication activities have steadily declined since 1990 when a record 1.5 million acres were treated. That trend continued last spring with treatment of 352,692 acres in 13 states, the lowest number since 1981. This decline coincides with the occurence of *E. maimaiga* throughout the area generally infested by gypsy moth.

In almost every State, the story was similar. The gypsy moth fungus was observed to be active just about everywhere gypsy moth caterpillars could be found.

New Jersey.—The decrease in gypsy moth defoliation levels was due to increased spray efforts and the widespread occurrence of the fungus disease. The impact of *E. maimaiga* was most obvious in the northern half of the State where, in past years, a typical gypsy moth outbreak would last for two or three years. It appears that north Jersey gypsy moth populations have a sudden buildup, but die off in the same year due to the fungus. In the southern half of the State, the fungus impacts are more sporadic. However, gypsy moth populations in southern Jersey tend to spread to new areas the year after infection by the fungus. If this pattern continues into the future, the days of massive 100,000+ acres of gypsy moth defoliation may be over in New Jersey. *(Source: John D. Kegg, New Jersey Department of Agriculture.)*

Delaware.—The gypsy moth fungus disease, that dramatically appeared in 1995, was able to rapidly spread and infect caterpillars at an earlier stage of development in 1996. Many caterpillars died when they were still small, resulting in less feeding damage. The number of complaints from people are dramatically lower than last year. Field surveys during last May and June indicated very low larval populations and only a few areas with noticeable defoliation. The southeastern portion of the State, where the gypsy moth populations only recently built up to defoliating levels, is the only area with noticeable populations of gypsy moth. *(Source: Donald A. Eggen, Delaware Department of Agriculture.)*

Michigan.—Gypsy moth populations in the central counties of the lower peninsula have been on a decline for the past four years and remain so this year. The fungal pathogen, *E. maimaiga*, was introduced into Michigan in 1991 and by 1996 had been documented in 28 counties surrounding the collapsed central infestation. The tenacious hold gypsy moth has had in Michigan seems to be weakening. A cool spring held back development of hatching larvae, while the ensuing wet weather seemed ideal for the fungus. Some tree trunks were reported as "hairy" from masses of dead 4th and 5th instar larvae. *(Source: Ronald J. Priest, Michigan Department of Agriculture.)*

Virginia.—*E. maimaiga* was experimentally released in Virginia beginning in 1990 and became obvious in gypsy moth populations in 1992. The first localized gypsy moth mortality, apparently resulting from the fungus, was seen at many sites across northern Virginia in 1994. Evidence of the fungus was

found in virtually all gypsy moth populations inspected in Virginia in 1995. The presence of the fungus was even confirmed in extremely low density gypsy moth populations in the central Piedmont area. A widespread population collapse was observed in all counties in northern Virginia in 1995. The acres treated in 1996 under the Virginia Cooperative Gypsy Moth Suppression Program was 89 percent less than treated in 1995. Field personnel attribute most of the decline in treatments, as well as the dramatic reduction in defoliation from a record 850,000 acres in 1995 to zero this summer, to the presence of the fungus. We are not sure what long-term impact the fungus will have on gypsy moth, but expect insect populations in some areas to rebound in the future. *(Source: Gary McAninch, Virginia Department of Agriculture and Consumer Services.)*

Pennsylvania.—According to qualitative and empirical data collected annually over the past several years, *E. maimaiga* has had a pronounced impact on gypsy moth populations. The fungus apparently is the primary natural mortality causal agent. As a result, these populations have undergone a general decline across most sections of the Commonwealth. This, in turn, has resulted in fewer acres being submitted for treatment during those years. Based upon previous outbreak episodes, we were expecting a resurgence of gypsy moth beginning in 1995. This did not materialize nor did the insect manifest itself in 1996. However, we feel that this situation is very fortuitous brought about by an unusual combination of environmental conditions favoring fungus development during the past several gypsy moth larval growing seasons. Control of these environmental conditions are, of course, beyond our ability to manage. As a result, we fully expect to see future buildups of the gypsy moth along with the demand for suppression programs in years ahead when we have dry, warm spring weather. With the introduction of *E. maimaiga* into the equation, these buildups, unfortunately, have become even more difficult to forecast. *(Source: Larry D. Rhoads, Pennsylvania Department of Conservation and Natural Resources.)*

Overall.--There is general consensus among scientists and pest managers that *E. maimaiga* is probably responsible for the decline in gypsy moth outbreaks and damage over the last few years. However, we do not know if this level of fungus activity will continue because *E. maimaiga* has been highly variable and unpredictable. This poses a dilemma for pest managers because treatment projects must be planned and carried forward well before it is known how active the fungus might be during the next gypsy moth season. Consequently, the use of environmentally safe and effective insecticides will continue to be important tools to reduce damage caused by gypsy moth outbreaks.

In closing, several points are emphasized:

* It is important to continue studies to fully understand how *E. maimaiga* will affect gypsy moth populations in the long term. This fungus is a recent member of a group of natural control agents that regulate gypsy moth populations in North America. Some of these biotic agents, like invertebrate predators, parasites, and the gypsy moth virus, have been part of gypsy moth population dynamics since at least the turn of the century, and evolved specific roles in that cycle of regulating populations. We do not know where or how the fungus will fit into this cycle in the long term, or what effect it might have on these other natural control agents.

* Expect gypsy moth outbreaks and subsequent damage to forests and trees to continue as the insect continues its natural spread into uninfested areas. Newly infested areas historically have sustained repeated defoliation and damage. It will be necessary to maintain adequate technical and financial resources to monitor gypsy moth populations (and fungus levels when tools become available) and to protect forests and trees in these areas.

* Expect that people will continue to inadvertently move the gypsy moth to uninfested areas of the country, as the insect hitchhikes on outdoor household articles, nursery stock, Christmas trees, and other commodities. These isolated infestations will still need to be detected, monitored and eradicated to prevent the insect from becoming permanently established outside of the generally infested area. Exclusion activities will still need to be continued to prevent the introduction of gypsy moth strains, especially those characterized by female moth flight, from other parts of the world.

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The Gypsy Moth Pathogen, *Entomophaga maimaiga*, in the Field versus Laboratory

Ann E. Hajek[1], Linda Butler[2], Scott R. A. Walsh[3], Julie C. Silver[3], Fred P. Hain[4], Felton L. Hastings[4], Thomas M. Odell[5], and David R. Smitley[6]

The pathogenic fungus, *Entomophaga maimaiga,* was first reported in North American gypsy moth, *Lymantria dispar* (L.) in 1989. This larval pathogen caused epizootics during both 1989 and 1990 in gypsy moth populations across a range of densities from innocuous to defoliating. During 1991 and 1992, *E. maimaiga* was released in 41 small plots in Maryland, Pennsylvania, Virginia, and West Virginia and became established in the majority of plots. During 1992, *E. maimaiga* caused epizootics in most release plots and spread throughout much of the contiguous northeastern range of the gypsy moth.

Insect pathogens are increasingly being investigated for biological control, but environmental safety is pivotal to their use. Limited impact of entomopathogens on nontarget species is especially critical where exotic or genetically engineered pathogens are being considered for release. Laboratory bioassays are almost always used to evaluate the impact of entomopathogens on nontarget insects. However, as a caveat, it is common that entomopathogens can infect hosts in the laboratory that are never found infected in the field.

Because of the potential for continued use of *E. maimaiga* for biological control, extensive laboratory bioassays were conducted to test the host specificity of *E. maimaiga*. *E. maimaiga* has been found infecting only gypsy moth in Japan, where it is native. Bioassays conducted using an isolate of *E. maimaiga* collected in Japan in 1984, demonstrated that this pathogen infected only Lepidoptera. During 1992 and 1993, further laboratory bioassays were conducted using northeastern North American isolates of *E. maimaiga* against species of Lepidoptera native to West Virginia. Approximately 1/3 of the 78 species tested became infected although in general, infection levels were very low. Infection in > 50 percent of individuals tested was found only in 3 species of lymantriid and 1 species of sphingid. In contrast, there are no published records of *E. maimaiga* infections in North America in any host except gypsy moth.

When epizootics caused by *E. maimaiga* occur, the lower boles of trees are characteristically covered with cadavers of larval gypsy moths that are attached by their prolegs. It frequently has been noted that only gypsy moth larval cadavers are present on tree boles, although many other species of Lepidoptera occur sympatrically in these forest ecosystems. However, the lack of conspicuous, infected alternate hosts does not provide irrefutable information about the specificity of this pathogen in the field.

We report results of studies evaluating infection of species of Lepidoptera other than gypsy moth in the field. During 1994, nontarget lepidopteran larvae were collected during the gypsy moth larval season from locations where *E. maimaiga* was active. These data are compared with laboratory bioassay results to evaluate the congruence of host specificity in laboratory and field. However, our studies were complicated by the sympatric occurrence of *Entomophaga aulicae*, a Lepidoptera-specific, North American native fungal entomopathogen closely related to *E. maimaiga,* that does not infect gypsy moth larvae. These morphologically identical fungal species are both members of the *E. aulicae* species complex. Genomic DNA probes have been developed that reliably differentiate between these two sister species, and these probes were used to distinguish between these two fungi during this study. Our efforts included quantitative field sampling of gypsy moth larvae to confirm activity of *E. maimaiga,* while concurrently sampling potential alternate hosts.

Materials and Methods

Seven 0.01-ha plots in the George Washington National Forest, Virginia, where *E. maimaiga* was introduced in 1991 or 1992, were chosen for intensive sampling. At all sites, abundant *E. maimaiga* in gypsy moth populations had been documented in 1992. Therefore, we expected that *E. maimaiga* would be active to some extent at these sites in 1994. At each site, 50 trees within the plot and adjacent to it were burlap-banded before second instars were present.

Each site, each week, 30 gypsy moth larvae and as many nontargets as could be found were collected. Larvae were checked daily to detect mortality. Cadavers were then dissected to detect resting spores or viral occlusion bodies. Infections by *E. aulicae* species complex were identified further using DNA probes and bioassays. Other entomophthoralean species could be identified using morphological features.

Isolation and Bioassay of Fungi. For any alternate host larvae that died and from which entomophthoralean conidia were discharged, we attempted to isolate the fungal pathogen causing disease. Such procedures provided a test of the identity of *E. maimaiga*, because for all *E. aulicae* species isolates tested to date, only *E. maimaiga* can infect gypsy moth larvae. *Entomophaga maimaiga* was successfully isolated from all infected nontarget hosts, and bioassays were conducted

using these isolates. Genomic DNA was extracted from all fungal samples and tested using DNA probes specific to *E. aulicae* and *E. maimaiga.*

Results

At George Washington National Forest, VA, *E. maimaiga* was active at all sites. Season-long gypsy moth infection levels varied from 40.8-97.5 percent. No gypsy moth larvae were infected by *E. aulicae.* In total, 6 of 1,511 lepidopteran larvae other than gypsy moth, belonging to 52 species in 7 families were collected from these plots. *E. maimaiga* was not found in any of the nontarget lepidopteran larvae collected at five of the sites, although infection was abundant in gypsy moth larvae. At each of the remaining two sites, only one alternate host was infected by *E. maimaiga*, yielding total infection levels of 0.3 percent for the forest tent caterpillar, *Malacosoma disstria*, and 1.0 percent for *Catocala ilia.* DNA probes and bioassays confirmed that both of these cadavers were positive for *E. maimaiga.*

The two sites where *M. disstria* and *C. ilia* were found infected by *E. maimaiga* did not have the highest levels of *E. maimaiga* infection among gypsy moth larvae, nor did they contain the most dense gypsy moth populations. In fact, there was no association between gypsy moth egg mass density and season-long infection by *E. maimaiga.* High levels of infection occurred across all densities.

Discussion

Of the 1,790 field-collected lepidopteran larvae reared during this study, only two individuals were infected by *E. maimaiga.* This was especially surprising for sites in the George Washington National Forest, where *E. maimaiga* concurrently caused heavy infections in gypsy moth larval populations. In plots where epizootics occurred in gypsy moth populations, our knowledge of the epizootiology of this disease suggests strongly that resting spores were germinating in the soil and infecting larvae; airborne conidia, actively ejected from newly dead hosts, were causing secondary infections. Therefore, nontarget Lepidoptera might have become infected by either spore type. Curiously, the two individual nontarget larvae that became infected were collected prior to the period of peak infection among gypsy moth larvae; none were collected during the period of peak infection of gypsy moth larvae.

Only 20 species of Lepidoptera that we collected in the field had been tested in the laboratory for susceptibility to *E. maimaiga.* Of these, six species became infected in the laboratory but only one of these, *M. disstria*, also was infected in the field. This species was infected at much higher levels in the laboratory than in the field.

Our study clearly demonstrates that information on host specificity based on laboratory bioassays would predict a broader host range and more abundant infection than was observed in the field. Based on information from our study, biological control practitioners should accept laboratory host range data as a liberal estimate of the environmental safety of insect pathogens. When possible, a more accurate determination of host range should be determined through actual field studies which take into account the frequently unknown interactions between hosts and pathogens that are not included in laboratory studies.

In conclusion, results from this study do not agree with results from laboratory bioassays, yet laboratory bioassay results are most frequently used to judge the environmental safety of fungal entomopathogens. Very few studies have been conducted that investigate the nontarget effects of fungal entomopathogens in the field. Our results demonstrate that host range in nature is clearly determined by unknown factors in addition to those generally studied in the laboratory suggesting that whenever possible, environmental safety of fungal entomopathogens should be investigated in the field as well as in the laboratory.

Note: Article adapted from: Ann E. Hajek, Linda Butler, Scott R. A. Walsh, Julie C. Silver, Fred P. Hain, Felton L. Hastings, Thomas M. Odell, and David R. Smitley. 1996. Host Range of the Gypsy Moth (Lepidoptera: Lymantriidae) Pathogen *Entomophaga maimaiga* (Zygomycetes: Entomophthorales) in the Field Versus Laboratory. Environmental Entomology, Vol. 25, No. 4, pp. 709-721.

See the original article for a complete list of references.

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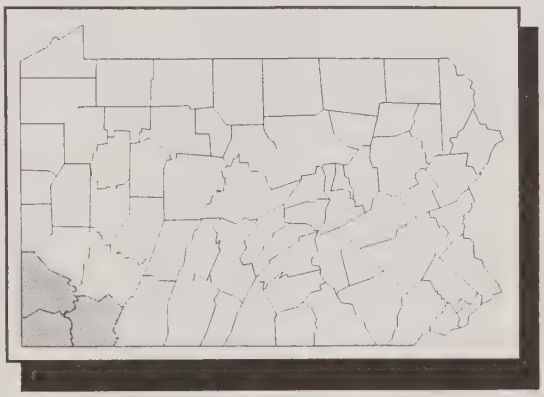
Studies of *Entomophaga maimaiga* in Pennsylvania

Barry B. Hunter

Entomophaga maimaiga (EM) was first observed in eastern Pennsylvania in 1989 and 1990. Because there were no naturally occurring populations of EM in the western part of the state in 1991, and the advancing front of the gypsy moth (GM) infestation was imminent, PA Bureau of Forestry (PBF) personnel wanted to introduce EM into selected forest areas in this part of the State. In the spring of 1991, California University of PA researchers, in cooperation with the PBF, established 17 experimental sites in Fayette, Greene, and Washington Counties



of southwestern PA. These sites were selected to determine whether EM could be established in forested areas where the fungus had not been observed. Several sites were identified and EM was introduced. Using two methods: (1) crushed EM cadavers, containing resting spores, were mixed into commercial potting soils; and (2) forest soils in which cadavers had fallen during the late summer of the previous year. At that time, no method had been developed to quantify the number of resting spores per cadaver; therefore, the amount of resting spores per experimental tree was unknown.

During the summer of 1992, a few early instar larvae containing resting spores were recovered from some of the sites and most from where they had been found in 1991. However, a majority of collected GM larvae (collections early in the summer) were reared to adults, and those that died were killed by insect parasites, other fungi, and by virus. But at two of the introduced sites, late summer collections, numerous late instar larvae were filled with resting spores. Resting spores, in fewer larvae, were also found at several other sites. This is important because these sites had low density larval populations, and the oaks and other tree species showed little or no feeding damage. During the summers of 1993 through 1996, the fungus increased dramatically and was found at all of the original 17 sites. As the percentage of EM-killed larvae increased, so did the total larval mortality resulting from all causes. Some of the sites were not observed in 1994 and 1995 because of low larval numbers. These same sites were studied in 1996 and larval numbers had increased, but the larvae found were EM-diseased or dead. No defoliation occurred within the sites, but adjoining forested areas showed significant feeding damage or massive defoliation.

The EM introduced areas had been protected and demonstrated that EM could be introduced into forested areas where egg masses were few, and that defoliating populations of the GM are not required for establishment and perpetuation of EM.

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Quantifying the Role of *Entomophaga maimaiga* in the Premature Collapse of a "Leading Edge" Gypsy Moth Population Near Lexington, VA, 1995 and 1996

R.E. Webb, G.B. White, K.W. Thorpe, and S.E. Talley

Introduction

Entomophaga maimaiga, a fungal pathogen native to Japan, has recently appeared in gypsy moth populations in the Eastern United States, often causing massive kill of late-season caterpillars. When conditions are right in the spring, resting spores produce a second type of spore (germ conidia) that young caterpillars pick up during daily travels. These germ conidia produce hyphae that grow vegetatively within the caterpillar and ultimately kill it. Hyphae grow through the outer wall of the insect and produce enormous numbers of secondary conidia that are transported by wind, thus spreading the infection to surrounding gypsy moth populations. These later-infected caterpillars produce mainly resting spores that overwinter in the soil and litter. Since this is an involved process that takes place over a number of weeks, most gypsy moth larvae die from the fungus as older caterpillars that have already done considerable feeding, and often a number of gypsy moths complete their development and so escape the fungus. Thus, it is still possible to have heavy defoliation despite impressive levels of fungus-induced mortality.

In 1993/1994, we monitored pathogen occurrence in gypsy moth populations in a number of woodlots near Lexington, Virginia, on the "leading edge" of the expanding North American gypsy moth infestation. The gypsy moth nuclear polyhedrosis virus (NPV) was found at significant levels only in woodlots with high gypsy moth populations, but *E. maimaiga* was recovered from woodlots with high or low gypsy moth densities. In 1995, we conducted studies in these woodlots in order to quantify the impact that the two diseases were having on gypsy moth populations as a function of their population density. These studies were continued in 1996. We hypothesized that *E. maimaiga* (fungus) should operate at lower gypsy moth populations densities than NPV (virus). The fungus should be considerably less density dependent than the virus, and due to different modes of action, the fungus and virus should operate independently of one another. The specific objectives of our 1995 study were: 1) to compute indices of impact for fungus and NPV; 2) to monitor the higher density plots to determine the onset of disease; and 3) to quantify early season disease levels and their correlation with the developing fungal and viral epizootics. A more general objective of this study was to quantify the impact of *E. maimaiga* in a "leading edge" gypsy moth population.

Ten woodlots near Lexington, Virginia, were chosen to reflect a range of gypsy moth population densities. Season-long total larval mortality (all sources) was calculated based on the number of larvae that died during the week after each collection. The following is an abbreviated account of our findings, with details to be published elsewhere.

Results

In 1995, levels of the fungus were high in all plots regardless of gypsy moth density, while virus levels were clearly higher in the high density plots than in the low density plots. In 1996, the virus was not a factor in the continued decline of the gypsy moth population in all plots, while fungal impact remained high. At the beginning of 1995, the highly susceptible, never-before-defoliated woodlots used in this study extended from the defoliating front (high density plots) into the "leading edge", where the (low density) plots were still several years away from serious defoliation. The populations in all woodlots fell dramatically without causing the high levels of defoliation that had been expected in the high density plots on the defoliating front, nor did the low density plots just within the leading edge undergo the normal progression from "release" to "progradation" to "culmination"; instead, gypsy moth populations went from low to non-detectable. The defoliating front that should have progressed through all plots simply disappeared. The gypsy moth is currently advancing southward and westward. The finding that *E. maimaiga* can indeed cause premature population collapse of gypsy moth populations in never-before-defoliated woodlots on the defoliating front and just within the leading edge may have profound implications for the future rate-of-spread in North America of this serious forest defoliator. This development should greatly aid the success of the "Slow the Spread" (STS) demonstration project.

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Introduction and Establishment of *Entomophaga maimaiga* in Michigan - 1991-1996

Leah Bauer and Dave Smitley

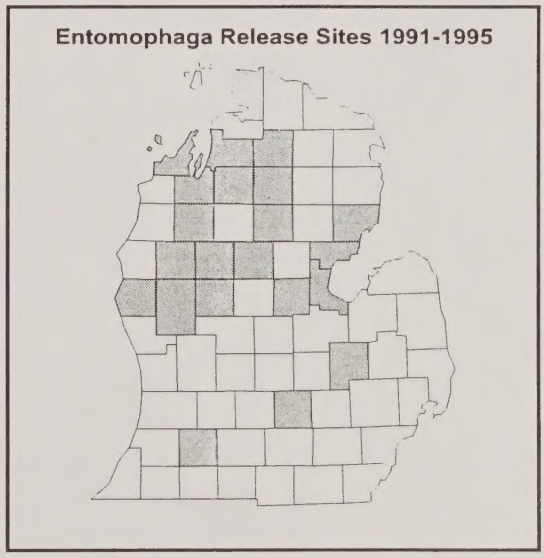
During the 1980's, a gypsy moth infestation expanded rapidly throughout the contiguous hardwood forests of Michigan's lower peninsula. This gypsy moth infestation, disjunct from the generally infested eastern U.S., lacked the diversity of parasites, predators, and pathogens which were introduced into the northeastern States over the last century.

In 1991, we surveyed gypsy moth infestations in Michigan's lower peninsula for prevalence of *E. maimaiga* in 11 counties. We also initiated field research in 3 counties to compare the efficacy of two inoculative-release methods: relocation of soil contaminated with resting spores vs. release of infected larvae. In addition, the Michigan Department of Natural Resources (DNR) set up operational introductions of *E. maimaiga* by placing resting spore-contaminated soil around individual trees in 20 sites in 9 counties. We made annual collections of larvae and cadavers, defoliation estimates, and egg mass counts at release sites and along transects to monitor the establishment and rate of spread of *E. maimaiga.*

The results of our gypsy moth pathogen surveys, experimental plots, and DNR release sites were: 1) *E. maimaiga* was probably not a cause of mortality in Michigan gypsy moth populations prior to 1991; 2) successful introduction of this fungus into gypsy moth infestations was readily achieved by placing infected gypsy moth caterpillars onto tree trunks, or by placing soil containing resting spores around the base of trees; 3) prevalence of infection and transmission rate were positively correlated with precipitation and relative humidity; and 4) fungal epizootics were responsible for reduced defoliation and lower egg mass counts.

In 1992, *E. maimaiga* was found in the 3 counties containing our research plots. The rate of fungal spread throughout Michigan during 1991-1992 was much lower than occurred in the eastern U.S., and may be explained by below normal rainfall during June when airborne conidia are released from gypsy moth cadavers. In June 1993, however, Michigan had twice the normal rainfall, and by the end of June, *E. maimaiga* was found in three additional counties across the middle of the lower peninsula. To facilitate operational spread of the fungus, gypsy moth coordinators were given cadavers containing resting spores collected from sites with *E. maimaiga* epizootics. These cadevers were placed on the ground in wooded areas in late summer in ca. 11 counties.

In 1994-1995, a new research site was established in southwestern Michigan; additionally, the prevalence of *E. maimaiga* in gypsy moth larvae and cadavers was determined in our 1991 research plots, the 1991 DNR release sites, and many of the original survey sites. The fungus spread to 13, 20, and 37 counties in 1994, 1995, and 1996, respectively. It is interesting to note that prior to the introduction of *E. maimaiga* in Michigan, defoliation had increased annually from 11 acres in 3 counties in 1979, to more than 700,000 acres in 45 counties by 1992. Gypsy moth populations have declined steadily since 1993, where defoliation fell to ca. 400,000 acres; by 1994 ca. 100,000 acres were defoliated. Although no single mechanism may be responsible, the initial collapse in 1993 appears correlated with high rainfall in June 1993 and subsequent spread of *E. maimaiga.* However, cold winter temperatures, implicated in overwintering egg mortality, may also be a factor in some localities. Another wet June in 1996 resulted in the expansive spread of *E. maimaiga* Statewide, and dramatic fungal epizootics. By 1996, only 3,200 acres of defoliation in two Michigan counties were detected. Based on 1995 egg mass counts, 42 of the 68 counties (62 percent) expected significant defoliation. However, no gypsy moth larvae or cadavers were found in 10 of those 42 counties (24 percent); and *E. maimaiga* was present in 57 percent of those counties, at an average prevalence of 15 percent.



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